# GENETIC, GROWTH, AND REPRODUCTIVE EFFECTS OF MICROWAVE RADIATION*

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THIS report reviews health aspects of nonionizing radiation exposure related to genetics, growth, and reproduction. Literature reporting effects in organisms is diverse and self-contradictory. This report neither analyzes nor attempts to resolve the conflicts which are included to the extent that they seem instructive.

Reproduction in mammals can be considered to begin at conception, when the genetic constitution of new individuals is determined. Normal growth includes the period of life between conception and sexual maturity. Our cultural traditions require special concern about our young, concern borne out, in part at least, by research evidence that developing, growing mammals are specially sensitive to external stressors. Microwaves are one such external stressor, and can affect growth and normal development. The influence does not appear to be strong.

## GROWTH

Effects of microwave exposure on growth have been determined by such endpoints as weight gain, congenital malformations, and resorptions. Exposure in the range 10-100 mW/cm.$^2$ during gestation decreased fetal weight in mice and rats.[1-4] Three studies[1-3] used multiple exposures involving most of the gestation days; the other[4] exposed animals only once or twice. Each study terminated development near the end of pregnancy, and the observations are presented in Table I.

Two studies of growing animals present opposite results. Exposure of juvenile mice[5] resulted in no observable effects on weight gain, but depression of growth has been reported in exposed chickens.[6,7]

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TABLE I. SUMMARY OF STUDIES OF ANIMAL WEIGHT EFFECTS OF *IN UTERO* EXPOSURE TO MICROWAVES

| *Reference* | *Animal* | *Exposure power density, mW/cm.²* | *Effect observed* |
|---|---|---|---|
| 1 | Mouse | 10 (daily during days 1-15 of gestation) | Decreased fetal weight |
| 2 | Mouse | 28 (daily during gestation) | Decreased fetal weight |
| 3 | Rat | 10 (daily on days 3-19 of gestation) | Relative decrease of body weight |
| 4 | Rat | 31* (absorbed dose rate once during interval, days 10-16 of gestation) | Decreased fetal weight |

*Absorbed dose rate, in mW/g. of body weight

TABLE II. SUMMARY OF STUDIES OF GROSS CONGENITAL MALFORMATIONS IN *IN UTERO*-EXPOSED ANIMALS

| *Animal* | *Exposure power density, mW/cm.²* | *Summary* | *Reference* |
|---|---|---|---|
| Mouse | 123 (estimated (1-time exposure) on gestation day 8 | Anomalies, notably exencephaly, dose-response relation suggested | 14, 15 |
| Mouse | 10 (daily on gestation days 1-15) | Increased frequency of developmental deviations | 1 |
| Mouse | 3.4-28 (daily during gestation) | Appearance of exencephaly in irradiated litters | 2 |
| Mouse and rat | 5-11 (18-306) hours) | Normal litters | 16 |
| Rat | 55-100* | Anomalies | 17, 18 |
| Rat | 10 (daily during gestation | No effects observed | 19 |
| Rat | 10, 40 (once on gestation day 9 or 16) | No adverse effects observed | 20 |
| Rat | 31* (once during gestation days 10-16) | Increased fetal resorptions | 4 |

*Absorbed dose rates, mW/g. of body weight

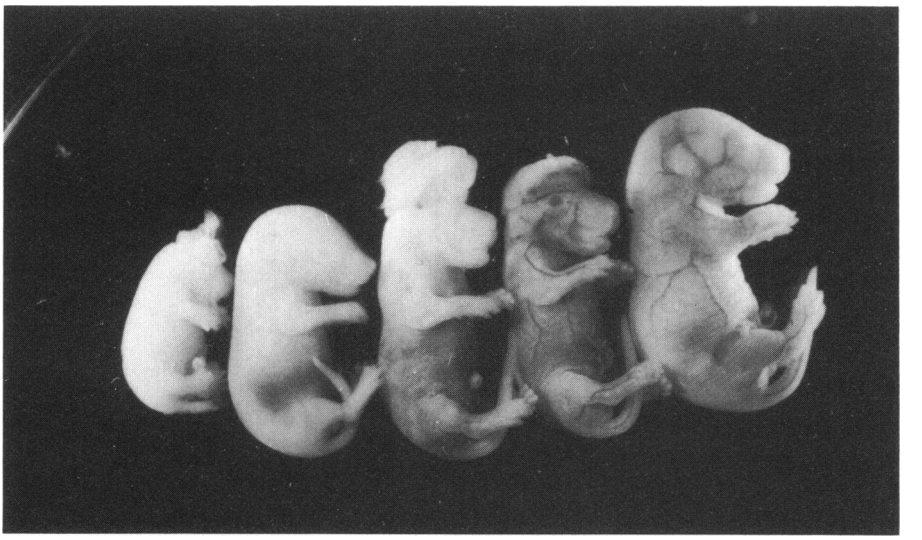

Fig. 1. Variety of anomalies in a single litter after microwave exposure (123 mW/cm.$^2$ estimated power density). From left to right: Dead fetus with reduced head (partial anencephaly); stunted fetus; normal size fetus with exencephaly; fetus with exencephaly and circulatory stasis; apparently normal fetus with circulatory stasis. In lower right, a resorption.

In humans, a case report of a radar repairman described weight loss[8] with episodes of tachycardia, venous thrombosis, and endocrine dysfunction. Not properly an effect on growth, the weight loss may reflect the kind of experience reported in studies of adult dogs[9-11] but not of adult rabbits.[12]

Congenital malformations have resulted from microwave exposure during early gestation, while the body organs are in early stages of formation. Animal studies of congenital anomalies are summarized in Table II. Figure 1 shows a litter from a mouse exposed on the eighth day of gestation, one of two peak periods of production of gross malformations in mice.[13] The figure shows exencephaly (brain hernia), stunting, resorptions, and apparently normal fetuses. The exposure, results of which are shown in the figure, and most of the exposures in Table II yielding positive effects, were sufficient to produce a heating effect in tissue. It is tempting to conclude that congenital malformations are a thermal response, except that the data also show malformations induced after repetitive exposures during gestation in mice.[2,3] The power densities used in these two studies are not clearly thermalizing.

TABLE III. SUMMARY OF ANIMAL STUDIES ON EFFECTS OF MICROWAVE EXPOSURE ON REPRODUCTIVE ORGANS

| *Animal* | *Exposure power density, mW/cm.²* | *Radiation frequency GHz.* | *Effect* | *Reference* |
|---|---|---|---|---|
| Mouse | 10 (2 hr daily for 5 or more months | 3 | Testicular* changes | 25 |
| Mouse | 50 (30-40 minutes 10 (100 minutes) | 1.7 | Altered* spermatogenesis | 26 |
| Mouse | 100 (4.5 min. 5 days per week for 59 weeks) | 9.27 (pulsed) | Testicular* degeneration | 23 |
| Mouse | 6.5 (total of 230 hours over 2-month period) | 2.45 | Testicular damage | 24 |
| Mouse | 400 (5 minutes) | ~ 10 (?) (3-cm waves) (pulsed) | Testicular lesions*, lesions of Graafian follicles, change in sexual cycle in females | 29 |
| Mouse | 0.344 (30 min.) 20-50 times prior to mating | ~ 10 (?) (3 cm. waves) | No deviations* from normal values for duration of pregnancy, number of offspring, or development of offspring of irradiated mothers | 32 |
| Rat | 250 (5-15 min.) | 24 | Testicular damage | 27 |
| Rat | 80 (10-80 min.) | 2.45 | Testicular damage | 28 |
| Rat | 0.1, 1.3 mW/cm.² (4 hr daily for 62-80 days | 2.98 (pulsed) | Change in sexual cycle in females | 30, 31 |

*Studies having data on irradiated animals' ability to sire offspring

A second aspect of malformations data is that positive data are derived mostly from mouse studies. Detailed criticism of experimental design, e.g., sample size, conception timing, timing of application of microwave stress, or statistical design and analysis, are beyond the scope of this report, and critical evaluation may indeed depend on additional data, particularly in systems known to respond after multiple exposures during appropriate periods in gestation.

Two different outcomes of pregnancy were reported among four women who received multiple diathermy treatments early in pregnancy.[21,22] Two spontaneous abortions ensued and two had normal deliveries. Three

women[21] received shortwave; one[22] received microwave diathermy. Current understanding of microwave effects on the embryo and fetus do not support a direct association between the exposure and the subsequent abortions.

## Reproduction

Animal studies suggest microwave interference with reproduction. Testicular changes have been reported in mice[23-26] and rats.[27,28] Lesions in Graafian follicles of mice were reported[29] associated with disturbances of the sexual cycle. Disturbance of sexual cycles was also observed in rats.[30,31] Studies of effects on reproductive organs are summarized in Table III. Among offspring of irradiated animals, increased stillbirths were detected in two studies,[25,29] and no effects on progeny were observed in two studies.[23,32] (The studies of Varma and co-workers will be summarized in the next section.)

As was the case with congenital malformations, reproductive effects have been reported mostly from studies of mice. Data also suggest the importance of multiple exposure.[25,30,31] However, two studies with multiple exposures showed no reproductive effects among offspring,[23,32] although the former study reported testicular damage. The range of power densities associated with effects detected among irradiated animals is 0.1-400 mW/cm.$^2$ The range associated with subsequent disturbance in litters is 10-400 mW/cm.$^2$; for no detected disturbances in litters the range is 0.344-100 mW/cm.$^2$

Testicular biopsy of one radar repairman demonstrated decreased spermatogenesis, tubular atrophy, focal necrosis, and interstitial edema. Over his four-year employment, exposures in excess of 30 W/cm.$^2$ were possible.[33]

## Genetic Effects

Genetic effects associated with point mutations should not occur as a result of microwave exposure because the photon energies are much too weak to ionize the genetic material, DNA. This expectation is borne out by research to date because effects are not demonstrated in tests of specific gene loci.[34] Other types of genetic change are possible, and include chromosomal structural changes, chromosomal number changes, and disturbances in gene function.

Chromosomal aberrations were produced by microwave exposure at power densities in the range of 5-5000 mW/cm.[2] [35-39] Some studies also showed chromosomal stickiness[40,41] and partial despiralization[35,41] of chromosomes. Structural aberrations may lead to dominant lethality *in utero*. Studies have shown dominant lethality in mice[42,43] and in *Drosophila* as a result of hyperthermia.[44] Chromosomal stickiness may interfere with the separation of chromosomes into daughter cells, and give rise to aneuploidy. Aneuploidy has been reported in tissue-culture cells chronically exposed to microwave radiation[45] and in *Drosophila*[44] following hyperthermia (38° C), in both cases due to the loss of a chromosome.

The Johns Hopkins study[46] referred to elsewhere in this volume is inconclusive about the relation between Down's syndrome among the offspring of radar workers and the workers' exposure to radar. In Down's syndrome, an extra chromosome is involved, and is considered diagnostic for the congenital disease. Other chromosomal changes may be observed in affected individuals, and include satellite association and formation of chromosome "rosettes" within mitotic cells. Both types of changes have been observed among irradiated animal cells (Leach, unpublished data).

Disturbances in gene function can be achieved by the timing or duration of application of stressors of biological systems during the production of a gene product. Temperature-sensitive gene loci serve as an example: a temperature shift during formation of the gene product results in a modified product that may affect the sensitivity and survivability of the cell. The production may be transient. Much of the microwave genetics reported by Webb[47] may be in the category of disturbed gene functions. In *E. coli,* effects on synthesis of a bacteriocidal product, colicin, were associated with microwave exposure.[48] Specific diseases have not thus far been associated with disturbances of gene function. In addition, perceived changes from transient disturbances of gene function may not be heritable.

The currently available genetic research in mammals, dominant lethality testing in mice,[42,43] demonstrates the erratic appearance of positive effects. The apparent inconsistency of the results is a source of concern, but it should be recognized that stage-specific effects during spermatogenesis could account for the variability. An additional complication is the recognition that either chromosomal changes or disturbed gene function can be the genetic mediators of the effects. To resolve questions affecting the genetic effects of microwave, additional research will be necessary.

## SUMMARY

Microwave exposure can, under circumstances not yet well enough specified to determine applicability to humans, cause congenital malformations and reduced reproductive capability. Exposure may also lead to chromosomal aberrations and anomalies of types associated with genetic diseases. However, attempts to establish the association have been inconclusive. It is suggested that point mutations are unlikely to result from microwave exposure. The importance of disturbances of gene function, however, is largely unknown, but such disturbances suggest a route to nonheritable genetic change within an individual.

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